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Case Report

Myocardial Infarction with Nonobstructive Coronary Arteries (MINOCA): What does an interventionist need to know?

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Abstract

In the 1980s, De Wood et al. reported that approximately 10% of patients with MI were found to have non-obstructive CAD. Currently, the prevalence may be even higher in the era of high-sensitivity cardiac troponin assays; because of the lower specificity to diagnose acute MI. MINOCA occurs in 5–15% of patients presenting with acute ST-segment elevation MI (STEMI) or non-ST segment elevation MI (NSTEMI). Many terms have been coined to describe patients with AMI or acute coronary syndrome (ACS) with normal or near-normal coronary arteries, such as MINOCA, MINCA (MI with normal coronary arteries) and INOCA (ischaemia and no obstructive coronary artery disease)

Keywords: CAD; high-sensitivity cardiac troponin assays; MINOCA; ST-segment elevation; acute coronary syndrome ; INOCA; AMI; obstructive coronary disease; hypertension; diabetes mellitus

Introduction

In the 1980s, De Wood et al. reported that approximately 10% of patients with MI were found to have non-obstructive CAD [1]. Currently, the prevalence may be even higher in the era of high-sensitivity cardiac troponin assays; because of the lower specificity to diagnose acute MI [2]. MINOCA occurs in 5–15% of patients presenting with acute ST-segment elevation MI (STEMI) or non-ST segment elevation MI (NSTEMI) [3]. Many terms have been coined to describe patients with AMI or acute coronary syndrome (ACS) with normal or near-normal coronary arteries, such as MINOCA, MINCA (MI with normal coronary arteries) and INOCA (ischaemia and no obstructive coronary artery disease) [4,5]. The term MINOCA is incorporated into the recently published Fourth Universal Definition of AMI [6]. Compared with obstructive coronary artery disease, factors associated with MINOCA include the female sex, younger age (<55 years), genetics and physiological stress [7]. A higher prevalence of MINOCA was found in younger patients (58.8% vs 61.3%, p<0.001), females (43% vs 24%, p<0.001), non-white patients (25% vs 12%, p<0.0001) and in patients presenting with non-STEMI (78% vs 51%, p<0.0001), compared to AMI with obstructive CAD [8-11]. Accurate diagnosis and subsequent management require the appropriate use of intravascular imaging and coronary function testing, in addition to echocardiographic and cardiac MRI (CMR) to assess for the presence of infarction or myocardial disorders without coronary involvement [12]. MINOCA is not a benign diagnosis, with outcomes similar to those of patients with acute MI and obstructive coronary disease up to 1 year (12-month mortality 0.6% versus 2.3%, respectively; p=0.68 [12]

Case Report

A 38-year-old man was referred to emergency department for ongoing chest pain. He had sudden onset of central, crushing chest pain for 7 hours, with severity increasing in the last 2 hours and the symptoms were associated with sweating. The patient had a known case of hypertension and was on medication for the last 2 years. The patient was also a pre-diabetic. His mother had hypertension and diabetes mellitus. There is no history of hypertension, diabetes mellitus or cardiovascular disease in his siblings.

General Examination: O2 saturation-95%, Pulse-110 bpm, Bp-110/90mmHg.Systemic Examination: no abnormality detected. Investigation: CBC, RFT, BSR, and Electrolytes were with in normal limits. ECG: ST elevation in infero-lateral leads. Cardiac Biomarkers: CPK-MB-82u/l and Trop 11.6u/l. Echo screening: hypokinetic inferior LV wall.

He was diagnosed as acute infero-lateral wall MI and was taken to the cath lab. His coronary angiography studies revealed normal coronary arteries. He was admitted in the CCU and was treated with Aspirin, Clopidogrel, LMWH, Atorvastatin, Beta-Blocker, Anxiolytics, PPI and Stool softener. On the following day, cardiac MRI was performed, which revealed curvilinear, confluent and patchy subendocardial enhancement noted in infero-posterior wall of the left ventricle. Features are compatible with myocardial infraction. He was conservatively managed and was discharged on 5th post MI day.

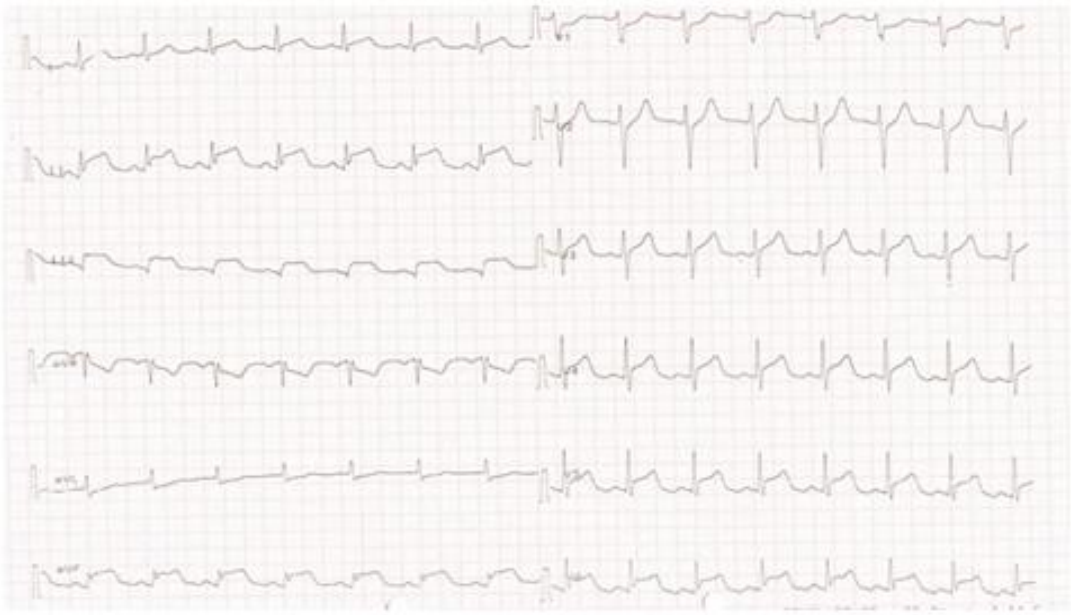

Figure 1. *ECG showing ST elevation in infero-lateral leads*

Definition and Pathophysiology of MINOCA

The diagnosis MINOCA requires (1) the presence of an AMI (according to the Fourth Universal Definition of AMI), (2) non-obstructive coronary arteries on invasive coronary angiography, defined as no coronary stenosis≥50% in any potential infarct-related artery, and (3) no clinically overt specific cause for the acute presentation [6]. In a patient presenting with symptoms of ischaemia, cardiac enzyme elevation and echocardiographic or electrocardiographic features suggestive of acute MI, a working diagnosis is made during angiography in the absence of culprit obstructive coronary artery disease (epicardial coronary artery stenosis ≥50%) or an apparent systemic cause for the presentation [13,14]. Approximately one-third of patients have been reported to present with suspected STEMI within an emergency setting and the remaining majority as NSTEMI patients undergoing subsequent angiography [3]. MINOCA disorders can be classified within the fourth universal definition of MI [6]. They may meet criteria for type 1 MI, where epicardial coronary artery disorders are diagnosed, or type 2 MI due to endothelial dysfunction or oxygen supply and demand mismatch, or myocardial injury [6]. (Figure. 2)

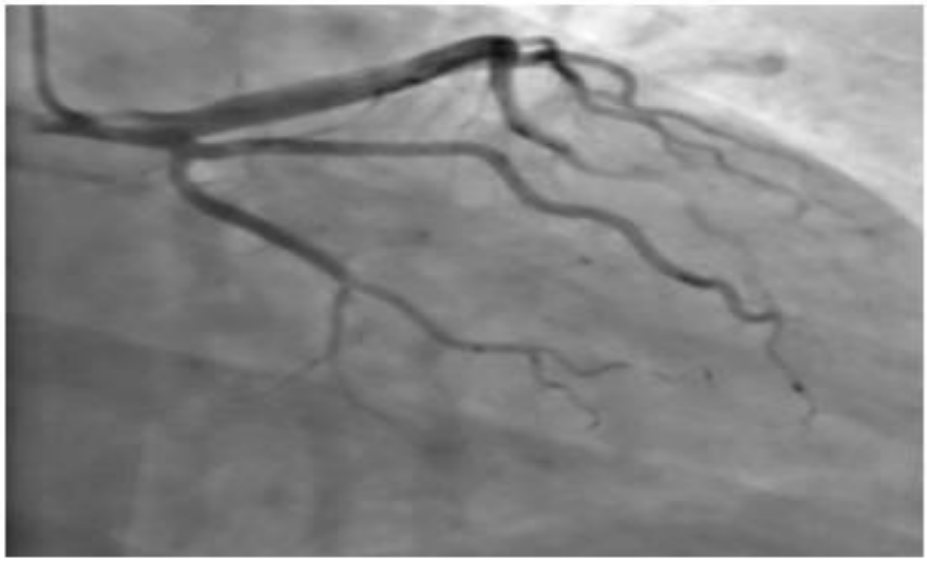

Figure 2. *Showing normal left coronary system*

Cardiac causes of MINOCA include:

1. Plaque disruption and plaque erosion
2. Spontaneous coronary artery dissection
3. Coronary artery spasm
4. Coronary micro vascular dysfunction
5, Coronary thrombus or embolism

Non-cardiac causes of MINOCA can result in myocardial injury include (PE), (end-stage) renal failure, sepsis; stroke and other forms of type 2 MI such as anemia and hyperthyroidism. They can also be associated with chest pain, elevated cardiac enzymes and ECG changes. (Figure. 3)

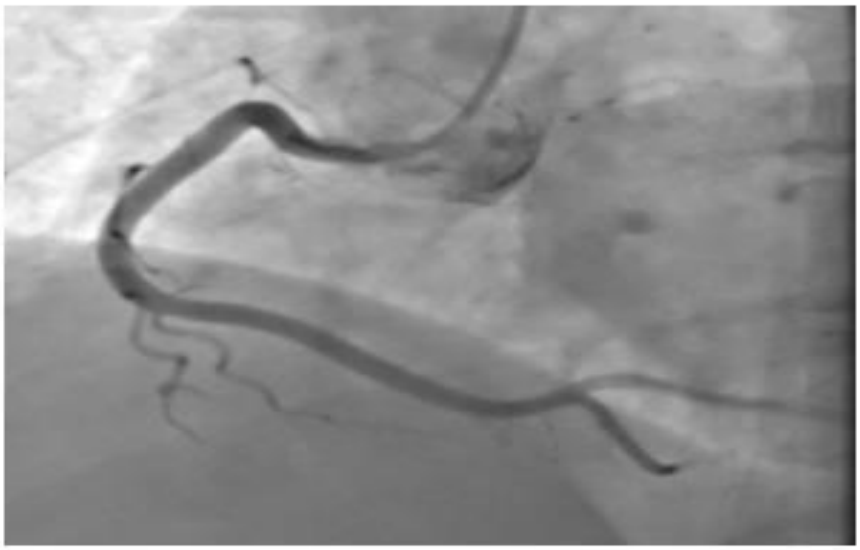

Figure 3. *Showing normal right coronary artery*

Discussion

Diagnosis and Evaluation of Patients with MINOCA

When a patient meets the criteria for a working diagnosis of MINOCA (universal acute MI criteria, infarct-related epicardial stenosis ≤50%, absence of overt alternative systemic cause) during angiography, then further invasive and adjunctive investigations should be considered at this point such as Coronary intravascular ultrasound (IVUS) or optical coherence tomography (OCT) [15,16,17]. Left ventriculography may also be of value in the assessment of other causes, such as takotsubo syndrome, and is routinely performed in many percutaneous coronary intervention (PCI) centers [18]. In addition to measurement of left ventricular end-diastolic pressure (LVEDP), ventriculography may also indicate an epicardial territorial distribution of impaired kinesis implicating a single epicardial artery, compared with a microvascular pattern involving an extended territory of one or more arteries. The upper limit of normal for LVEDP is 10 mmHg and LVEDP >18 mmHg is associated with an adverse post-MI prognosis [19]. Following invasive angiography, transthoracic echocardiography should be performed specifically assessing for the presence of regional wall motion abnormalities, embolic sources, pericardial effusion and typical features of takotsubo syndrome [20]. CMR can identify inflammation, oedema and scar and can assess myocardial function by T1 - and T2 -weighted imaging [21]. CMR is an important diagnostic tool and is guideline recommended in all patients with MINOCA [22]. If present on CMR, late gadolinium enhancement localizes the site of myocardial damage, and the pattern of distribution suggests the diagnosis [23]. Subendocardial or transmural enhancement is typically of ischaemic etiology. Subepicardial enhancement may be observed in myocarditis, cardiac sarcoid or cardiomyopathy associated with Duchenne muscular dystrophy. Mid-wall enhancement is associated with dilated cardiomyopathy, hypertrophic cardiomyopathy, Duchenne muscular dystrophy, Becker's muscular dystrophy, Anderson–Fabry disease, sarcoidosis or myocarditis. Finally, global endocardial enhancement is associated with amyloidosis, systemic sclerosis, hypereosinophilic syndrome or Churg–Strauss syndrome, whereas the absence of late gadolinium enhancement may be in keeping with microvascular dysfunction or a non-cardiac cause of the presentation [24]. CMR should be performed as soon as feasible after identification of MINOCA (within 4 weeks after hospital admission). However, in 8–67% of patients no abnormalities could be found, which leads to a therapeutic dilemma for clinicians [25]**.** (figure.4)

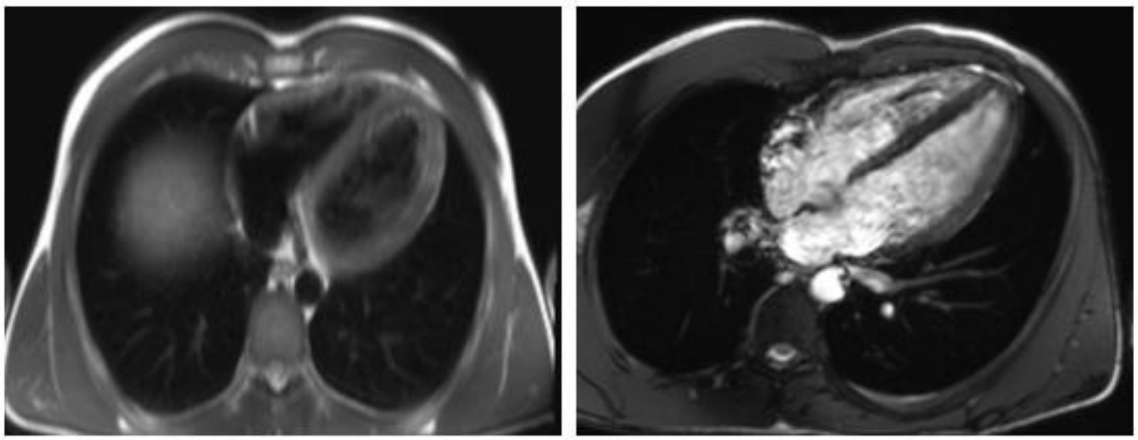

Figure 4. *CMR showing subendocardial enhancement noted in infero-posterior wall of the left ventricle*

Therapeutic Strategies for Patients with MINOCA

MINOCA secondary to plaque disruption or with evidence of ischaemic damage on CMR receive dual antiplatelet therapy (12 months followed by lifelong single agent), high-dose statin (including in patients with minimal plaque burden), β-blocker and ACEi or ARB [26, 27]. Mineralocorticoid receptor antagonists (MRA) may have a theoretical role in improving outcomes of MINOCA patients because aldosterone levels immediately after acute MI are associated with all-cause mortality. Aldosterone mediates the downstream effects of RAAS activation, including endothelial dysfunction, inflammation and fibrosis, but, at present, there are no trial data of MRA therapy in MINOCA patients [28].

Outcomes of Patients with MINOCA

With outcomes similar to those of patients with acute MI and obstructive coronary disease up to 1 year (12-month mortality 0.6% versus 2.3%, respectively; p=0.68), MINOCA is not benign [11]. Mortality and the incidence of major adverse cardiac events (MACE) for MINOCA patients are reported as comparable with those of patients with obstructive coronary artery disease, as and significantly worse than for the general population [29]. Within the SWEDEHEART registry, approximately one in four patients experience a MACE within 4 years, including death, recurrent MI, hospitalization with heart failure or ischemic stroke [30]. There are no studies focused on the effects of MINOCA on quality of life, including persistent ischaemic symptoms and psychosocial parameters. MINOCA-BAT will include a sub-study assessing the prevalence of angina pectoris in addition to health-related quality of life, anxiety, depression and psychiatric comorbidities [27].

Conclusion

MI with non-obstructive coronary arteries (MINOCA) is a heterogeneous working diagnosis requiring further investigation during and after invasive angiography. Clinicians should consider the use of intracoronary imaging and coronary physiology testing during angiography to assess for plaque disruption and vasospasticity. Cardiac MRI with gadolinium contrast is recommended in all MINOCA patients. MINOCA is not benign and has comparable outcomes with acute MI due to obstructive coronary artery disease. Treatment of the underlying cause is paramount although, at present, often empirical. There is an unmet clinical need for stratified therapy for patients with MINOCA.

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